Asian Research Journal of Mathematics
1(5): 1-12, 2016; Article no.ARJOM.30297

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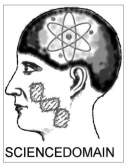

Mathematical Analysis of Effect of Isolation on the Transmission of Ebola Virus Disease in a Population

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Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/ARJOM/2016/30297
Editor(s):
(1) Dijana Mosic, Department of Mathematics, University of Nis, Serbia.
Reviewers:
(1) Molobe Ikenna Daniel, International Institute of Risk and Safety Management (Iirsm), Nigeria.
(2) Joel K. Weltman, Alpert Medical School, Brown University, USA.
(3) Edgar E. Escultura, V. Lakshmikantham Institute for Advanced Studies GVP College of Engineering, JNT University Kakinada, Vishakhapatnam, India.
Complete Peer review History: http://www.sciencedomain.org/review-history/17318

Original Research Article

Received: 31st October 2016
Accepted: 6th December 2016
Published: 23rd December 2016

Abstract

Outbreak of Ebola virus disease in early 2014 in West Africa is a major highlight for many researchers throughout the world because of the high mortality rate. Ebola disease is caused by a virus called the Ebola virus which can be transmitted from infected humans to uninfected humans through direct contact with the body fluids. Research has placed evidence that Ebola virus can be transmitted through the bodies of humans who recently died of the disease. Because of that, an epidemic model of $(S, E, I_u, I_d, I_s, R, D)$ is presented to study the dynamical spread of Ebola in the population. The existences of the disease free and unique endemic equilibrium were determined under certain conditions. Furthermore, the Local Stability analysis of the disease – free equilibrium (DFE) was investigated via the threshold parameter (Reproduction number R_0) obtained using the next generation matrix technique. The result shows that the DFE is asymptotically stable at Reproduction number less than unity $(R_0 < 1)$ and Unstable whenever Reproduction number is greater than unity $(R_0 > 1)$. Numerical simulations are carried out to confirm the analytical results and explore the possible behaviour of the formulated model. Numerical simulation shows that if the detection rate of infected undetected is sufficiently large, then the isolation techniques can lead to the eradication of the disease in the population.

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Keywords: Ebola; body fluids; reproduction number; $(S, E, I_u, I_d, I_s, R, D)$*; stability; isolation.*

1 Introduction

Ebola virus disease (EVD) formerly known as Ebola Haemorrhagic fever named after the river in Democratic Republic of Congo (formerly Zaire), is a severe, often fatal illness in human [1,2,3]. Ebola first appeared in 1976 in two simultaneous outbreaks in Nzara, Sudan, and in Yambuku, Democratic Republic of Congo [4,3]. The latter was in a village situated near the Ebola River, from which the disease takes its name. It is a virulent filo virus that is known to affect humans and primates. Ebola virus is 1 of 3 members of the filovirdae family (filo virus), along with genus Marburg virus and genus Cueva virus. Ebola virus comprises of 5 distinct species namely: Bundibugyo Ebola Virus (BDBV), Zaire Ebola virus (EBOV), Reston Ebola Virus (RESTV), Sudan Ebola Virus (SUDV) and Taiforest Ebola Virus (TAFV). BDBV, EBOV, and SUDV have been associated with large EVD outbreak in Africa, whereas RESTV and TAFV have not. The RESTV species found in Philippines and China Republic can infect humans, but no illness or death in humans from the species has been reported to date except BDBV. EBOV and SVDV which has same number of mortality rate [5,6].

Ebola was introduced into human population through close contact with the blood, secretions, organs or other bodily fluids of infected animals. In Africa, infection has been documented through the handling of infected chimpanzees, gorillas, fruit bats, monkeys, forest antelope and porcupines found ill or dead in the rainforest. It then spread in the community through human to human transmission, with infection resulting from direct contact (through broken skin or mucous membranes) with the blood, secretions, organs or other bodily fluids of infected people, and indirect contact with environments contaminated with such fluids [5,6]. The incubation period of this deadly disease is 2 – 21 days and infectious period is 4 -10 days [7]. Ebola is characterized by initial flu – like symptoms including "sudden onset of high fever greater than 38.6 degree Celsius or 101.5 degree Fahrenheit, fatigue, muscle pain, stomach pain, diarrhea sore throat, abdominal pain, unexplained hemorrhage and headache". This then rapidly progresses to vomiting of blood, rash, symptoms of impaired Kidney and Liver function, and in some cases it leads to both internal and external bleeding [5,8]. Most infected individuals die within 10 days of their initial infection [9,10]. Burial ceremonies in which mourners have direct contact with the body of the deceased person can also play a role in the transmission of Ebola [2,4].

In early 2014 outbreak in West Africa, it was reported that about 30% of infections were caused by a contact with the dead bodies that recently died of Ebola disease [2,11,12]. The number of dead bodies who recently died because of Ebola disease is related to the rate of the infection because of its burial process [2,13]. This evidence motivates us to explore the effect of burial process in a model of Ebola virus transmission by adding the dead compartment.

The threat posed by Ebola virus in human population initiated and prompted this research work to develop an epidemiological model that incorporated the dead individuals, infected undetected, infected detected and isolated individuals. There are two control measures that affect the transmission of Ebola virus. One form of the control is to accelerate the safe burial process of the bodies of people who recently died from the Ebola disease. This form of control is necessary due to the fact that the Ebola virus can survive in the dead bodies for 7 days [13]. The other control is to isolate infected humans who have been detected to reduce the transmission of Ebola virus from infected humans.

2 Model Formulation

A dynamical system consisting ordinary differential equation is used to construct the Ebola disease model in this article. We assume that the human population is divided into seven (7) compartments namely: susceptible (S), exposed (E), Infected undetected (I_u), Infected detected (I_d), Infected isolated (I_s), Recovered (R), and death individuals (D).

The susceptible population is increased by recruitment into the population at a rate π, this population is decreased by infection acquired from effective contact rate with infectious individuals in the Infected undetected , Infected detected and the infected dead individuals at a rate given by $(\beta_1, \beta_2, \beta_3)$ respectively; and finally reduced by the Natural death rate μ .

The fraction $(\beta_1 SI_u + \beta_2 SI_d + \beta_3 D)$ move to the Exposed individuals and the population is further reduced by σ which is the rate at which the exposed individuals are being isolated, κ which is the progression rate of the exposed individuals to the active Ebola stage, and by natural death rate μ .

The population of the infected undetected individuals is increased by the progression rate of exposed individuals κ , and finally reduced by γ_1 which is the rate of discovery of the undetected individuals by contact tracing, and (δ_u, μ) which is the disease induced rate of the undetected individuals due to the disease and by natural death rate respectively.

The undetected infected population is increased by the progression rate of exposed individuals κ , reduced by the rate at which the undetected individuals are being isolated due to facilities in the environment (γ_2) and finally decreased by the (δ_d, μ) which is the disease induced death rate of the detected individuals due to the disease and by natural death rate respectively.

Infected isolated population is increased by (σ, γ_2) which is the isolation rate of the exposed individuals and infected detected individuals respectively. The population was decreased by disease induced rate of isolated individuals (δ_s) , natural death rate (μ) , and the recovery rate of the isolated individuals (α).

The recovered population is increased by the recovery rate of the isolated individuals (α), and reduced by natural death rate (μ).

Finally, the infected dead individual is increased by the disease induced rate of infected undetected, infected detected and isolated individuals $(\delta_u, \delta_d, \delta_s)$. The population is reduced by the safe burial rate of the infected dead bodies (θ) .

In summary, the govern model is given by the system of differential equations below:

$$\frac{dS}{dt} = \pi - \beta_1 SI_u - \beta_2 SI_d - \beta_3 SD - \mu S$$

$$\frac{dE}{dt} = \beta_1 SI_u + \beta_2 SI_d + \beta_3 SD - \sigma E - \kappa E - \mu E$$

$$\frac{dI_u}{dt} = (1-\omega)\kappa E - \gamma_1 I_u - \delta_u I_u - \mu I_u$$

$$\frac{dI_d}{dt} = \omega\kappa E + \gamma_1 I_u - \gamma_2 I_d - \delta_d I_d - \mu I_d \qquad (1)$$

$$\frac{dI_s}{dt} = \sigma E + \gamma_2 I_d - \delta_s I_s - \alpha I_s - \mu I_s$$

$$\frac{dR}{dt} = \alpha I_s - \mu R$$

$$\frac{dD}{dt} = \delta_u I_u + \delta_d I_d + \delta_s I_s - \theta D$$

Table 1. Description of parameter of the model

Parameters	Description
π	Recruitment rate
β_2	Contact rate with undetected infected
β_2	Contact rate with detected Infected
β_3	Contact rate with dead bodies
μ	Natural death rate
σ	The rate at which exposed individuals are isolated due to contact tracing
κ	Progression rate of individuals in exposed stage to active Ebola
ω	Endogenous reactivation rate
γ_1	Detection rate for infected undetected Individuals
γ_2	The rate at which Infected detected individuals are Isolated
δ_u	Disease – induced death rate for undetected individuals
δ_d	Disease – induced death rate for detected individuals
δ_s	Disease – induced death rate for Isolated Infected Individuals
α	Recovery rate of isolated individuals
θ	Safe Burial rate

3 Analysis of the Model

3.1 Disease free equilibrium (DFE)

At equilibrium, (1) is set to be equal to zero.

That is:

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI_u}{dt} = \frac{dI_d}{dt} = \frac{dI_s}{dt} = \frac{dR}{dt} = \frac{dD}{dt} = 0 \tag{2}$$

The disease free equilibrium is obtained as,

$$(S, E, I_u, I_d, I_s, R, D) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0\right) \tag{3}$$

3.2 Endemic equilibrium point (EEP)

Besides the disease – free equilibrium point, we shall show that the formulated model (1) has an endemic equilibrium point. The endemic equilibrium point is a positive steady state solution where the disease persists in the population.

Setting $(\sigma+\kappa+\mu)=\phi_1,(1-\omega)=\phi_2,(\gamma_1+\delta_u+\mu)=\phi_3,(\gamma_2+\delta_d+\mu)=\phi_4,(\delta_s+\alpha+\mu)=\phi_5$ into equation (1) to have:

$$\frac{dS}{dt}=\pi-\beta_1 SI_u-\beta_2 SI_d-\beta_3 SD-\mu S$$

$$\frac{dE}{dt}=\beta_1 SI_u+\beta_2 SI_d+\beta_3 SD-E\phi_1$$

$$\frac{dI_u}{dt}=\phi_2\kappa E-I_u\phi_3$$

$$\frac{dI_d}{dt}=\omega\kappa E+\gamma_1 I_u-I_d\phi_4 \tag{4}$$

$$\frac{dI_s}{dt}=\sigma E+\gamma_2 I_d-I_s\phi_5$$

$$\frac{dR}{dt}=\alpha I_s-\mu R$$

$$\frac{dD}{dt}=\delta_u I_u+\delta_d I_d+\delta_s I_s-\theta D$$

We define $(S,E,I_u,I_d,I_s,R,D)=(S^{**},E^{**},I^{**}{}_u,I^{**}{}_d,I^{**}{}_s,R^{**},D^{**})$ and set (4) to be equal to zero. Thus,

$$S^{**}=\frac{\phi_1}{(\beta_1 A+\beta_2 C+\beta_3 H)} \tag{5}$$

$$E^{**}=\frac{\pi}{\phi_1}-\frac{\mu}{(\beta_1 A+\beta_2 C+\beta_3 H)} \tag{6}$$

$$I_u{}^{**}=\frac{\pi\phi_2\kappa}{\phi_1\phi_3}-\frac{\phi_2\kappa\mu}{\phi_3(\beta_1 A+\beta_2 C+\beta_3 H)} \tag{7}$$

$$I_d^{**} = \frac{\pi(\omega\kappa\phi_3 + \gamma_1\phi_2\kappa)}{\phi_1\phi_3\phi_4} - \frac{\mu(\omega\kappa\phi_3 + \gamma_1\phi_2\kappa)}{\phi_3\phi_4(\beta_1 A + \beta_2 C + \beta_3 H)} \tag{8}$$

$$I_s^{**} = \frac{(\sigma\phi_3\phi_4 + \gamma_2\omega\phi_3\kappa + \gamma_1\gamma_2\phi_2\kappa)}{\phi_3\phi_4\phi_5}\left[\frac{\pi}{\phi_1} - \frac{\mu}{(\beta_1 A + \beta_2 C + \beta_3 H)}\right] \tag{9}$$

$$R^{**} = \frac{\alpha(\sigma\phi_3\phi_4 + \gamma_2\omega\phi_3\kappa + \gamma_1\gamma_2\phi_2\kappa)}{\phi_3\phi_4\phi_5\mu}\left[\frac{\pi}{\phi_1} - \frac{\mu}{(\beta_1 A + \beta_2 C + \beta_3 H)}\right] \tag{10}$$

$$D^{**} = \frac{1}{\theta\phi_3}\left[\delta_u\phi_2\kappa + \frac{\delta_d(\omega\kappa\phi_3 + \gamma_1\phi_2\kappa)}{\phi_4} + \frac{\delta_s(\sigma\phi_3\phi_4 + \gamma_2\omega\phi_3\kappa + \gamma_1\gamma_2\phi_2\kappa)}{\phi_4\phi_5}\right]\left[\frac{\pi}{\phi_1} - \frac{\mu}{(\beta_1 A + \beta_2 C + \beta_3 H)}\right] \tag{11}$$

Where:

$$\frac{\phi_2\kappa}{\phi_3} = A,\ \frac{\omega\kappa\phi_3 + \gamma_1\phi_2\kappa}{\phi_3\phi_4} = C,\ \frac{1}{\theta\phi_3}\left[\delta_u\phi_2\kappa + \frac{\delta_d(\omega\kappa\phi_3 + \gamma_1\phi_2\kappa)}{\phi_4} + \frac{\delta_s(\sigma\phi_3\phi_4 + \gamma_2\omega\phi_3\kappa + \gamma_1\gamma_2\phi_2\kappa)}{\phi_4\phi_5}\right] = H$$

3.3 Basic reproduction number

In many mathematical model of infectious disease, Basic reproduction number plays an important role to describe qualitative analysis of the model. The basic reproduction number (R_0) measures the average number of secondary infected individual generated in his or her infectious period in the population of Susceptible [5,14]. If $R_0 < 1$, then the disease dies out and spread whenever it exceeds unity i.e $(R_0 < 1)$. Using the Next generation matrix, the non – negative matrix F of the new infection terms (Transmission) and the non – singular matrix V of the other remaining transfer terms (Transition) are given by FV^{-1}:

Where:

$$F = \begin{pmatrix} 0 & \frac{\beta_1\pi}{\mu} & \frac{\beta_2\pi}{\mu} & 0 & \frac{\beta_3\pi}{\mu} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \qquad V = \begin{pmatrix} P_1 & 0 & 0 & 0 & 0 \\ -P_2 & P_3 & 0 & 0 & 0 \\ -\omega\kappa & -\gamma_1 & P_4 & 0 & 0 \\ -\sigma & 0 & -\gamma_2 & P_5 & 0 \\ 0 & -\delta_u & -\delta_d & -\delta_s & \theta \end{pmatrix} \tag{12}$$

Where: $P_1 = (\sigma + \kappa + \mu), P_2 = (1-\omega)\kappa, P_3 = (\gamma_1 + \delta_u + \mu), P_4 = (\gamma_2 + \delta_d + \mu), P_5 = (\delta_s + \alpha + \mu)$

The basic reproduction number, $R_0 = \rho(FV^{-1})$, is the spectral radius of the product FV^{-1}. Hence, for the model (1), we arrive at:

$$R_0 = \frac{\pi\left(\beta_1 P_2 P_4 P_5 \theta + \kappa\omega\theta P_3 P_5 \beta_2 + \theta P_2 P_5 \beta_2 \gamma_1 + \kappa\omega P_3 P_5 \beta_3 \delta_d + \kappa\omega P_3 \beta_3 \delta_s \gamma_2 + \sigma P_3 P_4 \beta_3 \delta_s + P_2 P_4 P_5 \beta_3 \delta_u + P_2 P_5 \beta_3 \delta_d \gamma_1 + P_2 \beta_3 \delta_s \gamma_1 \gamma_2\right)}{\mu P_1 P_3 P_4 P_5 \theta}$$

3.4 Local stability of the model

Using the basic reproduction number obtained for the model (1), we analyze the stability of the equilibrium point in the following result.

Theorem: The disease – free state, is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ [14].

Proof: The Jacobian matrix of the system (1) evaluated at the disease – free equilibrium point, obtained as

$$J = \begin{pmatrix} -\mu & 0 & -\frac{\beta_1\pi}{\mu} & -\frac{\beta_2\pi}{\mu} & 0 & 0 & -\frac{\beta_3\pi}{\mu} \\ 0 & -P_1 & \frac{\beta_1\pi}{\mu} & \frac{\beta_2\pi}{\mu} & 0 & 0 & \frac{\beta_3\pi}{\mu} \\ 0 & -P_2 & -P_3 & 0 & 0 & 0 & 0 \\ 0 & \omega\kappa & \gamma_1 & -P_4 & 0 & 0 & 0 \\ 0 & \sigma & 0 & \gamma_2 & -P_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -\mu & 0 \\ 0 & 0 & \delta_u & \delta_d & \delta_s & 0 & -\theta \end{pmatrix} \tag{13}$$

Where: $P_1 = (\sigma + \kappa + \mu), P_2 = (1-\omega)\kappa, P_3 = (\gamma_1 + \delta_u + \mu), P_4 = (\gamma_2 + \delta_d + \mu), P_5 = (\delta_s + \alpha + \mu)$

We need to show that all the eigenvalues of J are negative. The eigenvalues of the matrix J are the roots of the characteristic equation

$$\lambda^7 + r_1\lambda^6 + r_2\lambda^5 + r_3\lambda^4 + r_4\lambda^3 + r_5\lambda^2 + r_6\lambda + r_7 = 0 \tag{14}$$

Where

r_i (for $i = 1, 2...6$) are representative of some expression

$$r_7 = \pi\beta_1 P_2 P_4 P_5 \theta + \pi\kappa\omega\theta P_3 P_5 \beta_2 + \pi\theta P_2 P_5 \beta_2 \gamma_1 + \pi\kappa\omega P_3 P_5 \beta_3 \delta_d + \pi\kappa\omega P_3 \beta_3 \delta_s \gamma_2 + \pi\sigma P_3 P_4 \beta_3 \delta_s + \pi P_2 P_4 P_5 \beta_3 \delta_u + \pi P_2 P_5 \beta_3 \delta_d \gamma_1 + \pi P_2 \beta_3 \delta_s \gamma_1 \gamma_2 - \mu\theta P_1 P_3 P_4 P_5$$

Employing the Descartes' rule of signs [15], which states that all roots of polynomial (14) have negative real part and distinct, if and only if the coefficient r_i are negative for $i = 1, 2, 3, 4, 5, 6, 7$.

Hence, it is *locally Asymptotically stable* if $r_7 > 0$:

Such that:

$$\left(\begin{array}{l}\pi\beta_1 P_2 P_4 P_5\theta + \pi\kappa\omega\theta P_3 P_5\beta_2 + \pi\theta P_2 P_5\beta_2\gamma_1 + \pi\kappa\omega P_3 P_5\beta_3\delta_d + \pi\kappa\omega P_3\beta_3\delta_s\gamma_2 + \pi\sigma P_3 P_4\beta_3\delta_s + \\ \pi P_2 P_4 P_5\beta_3\delta_u + \pi P_2 P_5\beta_3\delta_d\gamma_1 + \pi P_2\beta_3\delta_s\gamma_1\gamma_2 - \mu\theta P_1 P_3 P_4 P_5\end{array}\right) > 0 \quad (15)$$

Further simplification in terms of reproduction number yields

$$\frac{\pi\left(\beta_1 P_2 P_4 P_5\theta + \kappa\omega\theta P_3 P_5\beta_2 + \theta P_2 P_5\beta_2\gamma_1 + \kappa\omega P_3 P_5\beta_3\delta_d + \kappa\omega P_3\beta_3\delta_s\gamma_2 + \sigma P_3 P_4\beta_3\delta_s + P_2 P_4 P_5\beta_3\delta_u + P_2 P_5\beta_3\delta_d\gamma_1 + P_2\beta_3\delta_s\gamma_1\gamma_2\right)}{\mu P_1 P_3 P_4 P_5\theta} < 1 \quad (16)$$

Equation (16) implies $R_0 < 1$

Therefore, all the eigenvalues of the Jacobian matrix J have negative real parts when $R_0 < 1$, hence the disease-free equilibrium point is locally Asymptotically stable.

4 Numerical Results

In this phase, we study numerically the expression and behaviour of the system (1) employing some of the parameter values compatible with Ebola [5,13] as given in Table 2 and by considering the initial conditions, $S(0) = 1000,\ E(0) = 500,\ I_u(0) = 300,\ I_d(0) = 250,\ I_s(0) = 150,\ R(0) = 100,\ D(0) = 20.$

The numerical simulations are evaluated using the Runge-Kutta order 4 embedded in mathematical software (Maple 18). The results (figures) of the numerical solutions are given in the Figs. 1 – 4.

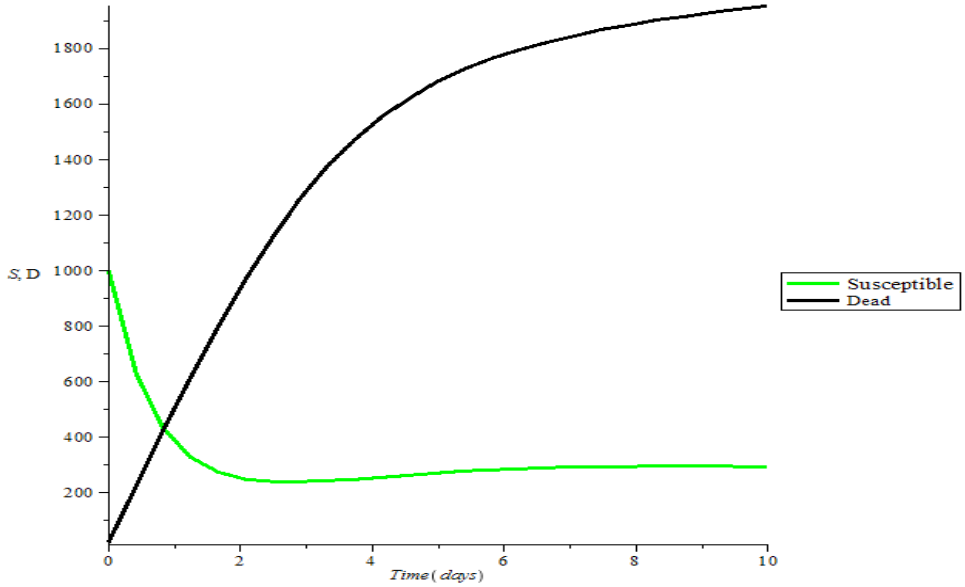

Fig. 1. The graph of (S, D) population against time where:

$\beta_1 = 0.000118, \beta_2 = 0.000118, \beta_3 = 0.000118, \mu = 0.02, \sigma = 0.2, \kappa = 0.6, \omega = 0.03, \gamma_1 = 0.12,$
$\gamma_2 = 0.12, \delta_u = 0.937, \delta_d = 0.937, \delta_s = 0.937, \alpha = 0.225, \theta = 0.2$

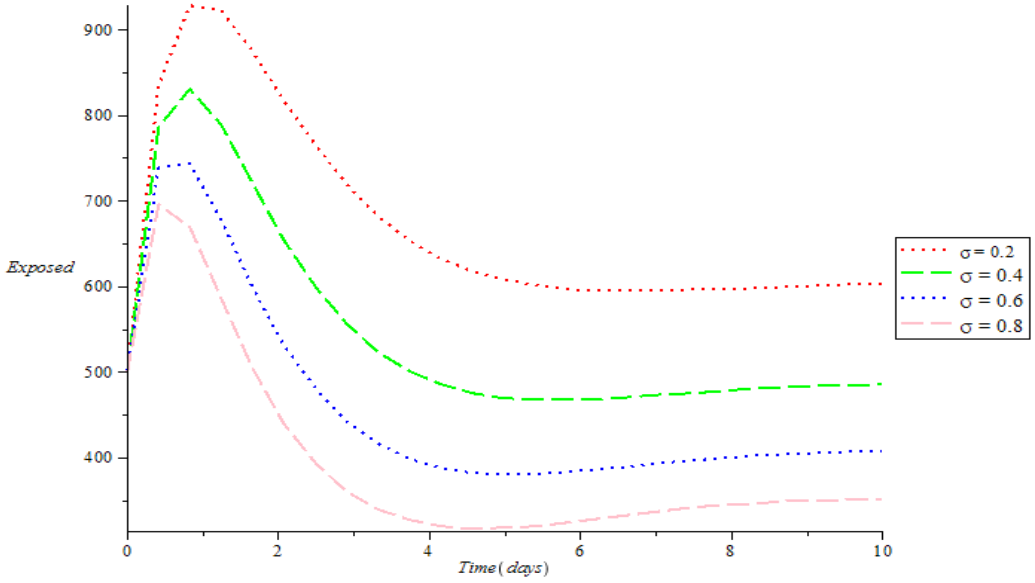

Fig. 2. The graph of varried exposed population against time where:

$\beta_1 = 0.000118, \beta_2 = 0.000118, \beta_3 = 0.000118, \mu = 0.02, \sigma = 0.2...0.8, \kappa = 0.6, \omega = 0.03, \gamma_1 = 0.12,$
$\gamma_2 = 0.12, \delta_u = 0.937, \delta_d = 0.937, \delta_s = 0.937, \alpha = 0.225, \theta = 0.2$

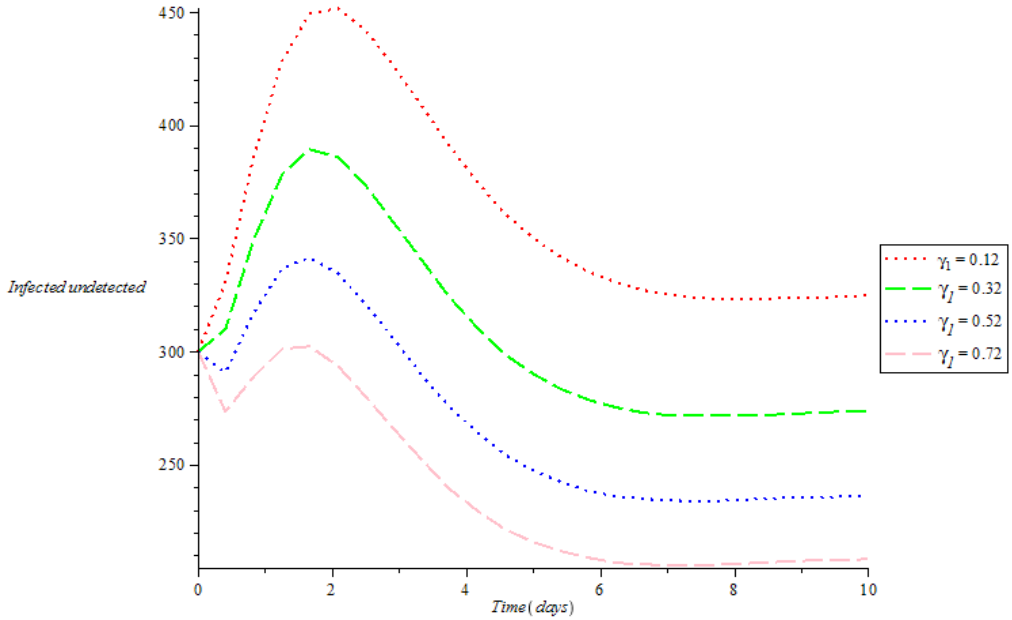

Fig. 3. The graph of varried Infected undetected population against time where:

$\beta_1 = 0.000118, \beta_2 = 0.000118, \beta_3 = 0.000118, \mu = 0.02, \sigma = 0.2, \kappa = 0.6, \omega = 0.03, \gamma_1 = 0.12...0.72,$
$\gamma_2 = 0.12, \delta_u = 0.937, \delta_d = 0.937, \delta_s = 0.937, \alpha = 0.225, \theta = 0.2$

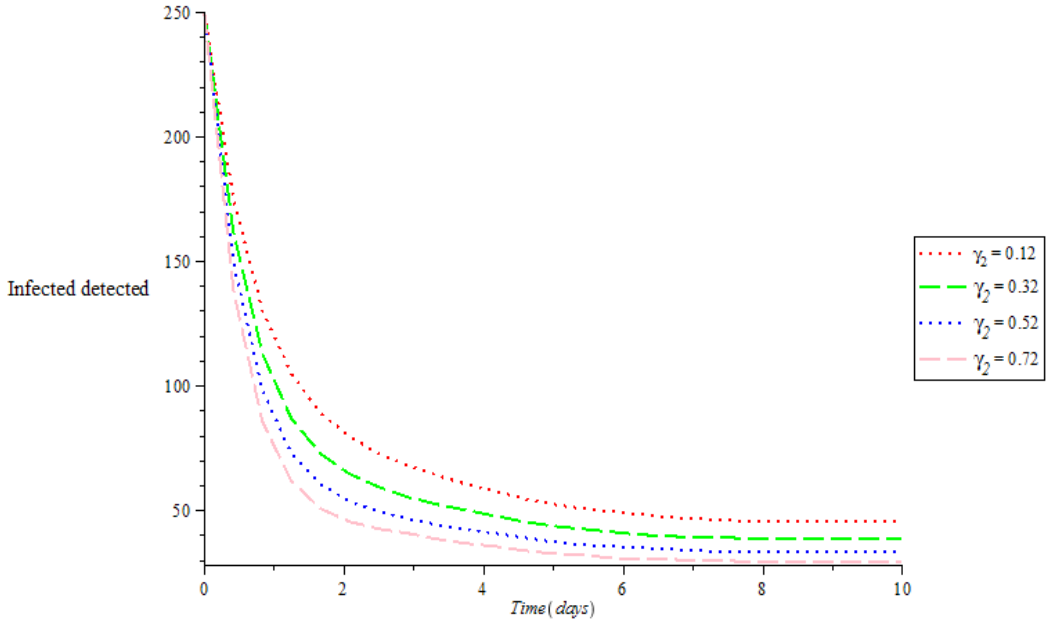

Fig. 4. The graph of varried Infected detected population against time where:
$\beta_1 = 0.000118, \beta_2 = 0.000118, \beta_3 = 0.000118, \mu = 0.02, \sigma = 0.2, \kappa = 0.6, \omega = 0.03, \gamma_1 = 0.12,$
$\gamma_2 = 0.12...0.72, \delta_u = 0.937, \delta_d = 0.937, \delta_s = 0.937, \alpha = 0.225, \theta = 0.2$

Table 2. Baseline parameters and values used in simulation

Parameters	Values	Reference
π		Assumed
β_1	0.000118	Assumed
β_2	0.000118	Assumed
β_3	0.000118	Assumed
μ	0.02	[5]
σ	0.2	[5]
κ	0.6	[5]
ω	0.03	[5]
γ_1	0.12	[5]
γ_2	0.12	[5]
δ_u	0.937	[7]
δ_d	0.937	[7]
δ_s	0.937	[7]
α	0.225	Assumed
θ	0.2	[5]

5 Discussion and Conclusion

An epidemic model of $(S, E, I_u, I_d, I_s, R, D)$ is presented to study and analyzed to gain more insight into the dynamical spread of Ebola in the population. Furthermore, the research shows that the disease free equilibrium is locally asymptotically stable if the threshold parameter (Reproduction number R_0) obtained using the next generation matrix technique is less than unity $(R_0 < 1)$ and Unstable whenever Reproduction number is greater than unity $(R_0 > 1)$. The model shows the danger posed by infected individuals who have not been detected and a need to improve the strategy for detecting the infected individuals.

Numerical solution shows the following results:

Fig. 1: Shows the graph of (S, D) population as time increases.

Fig. 2: Shows the effect of σ in the exposed population. It shows that when the isolation of Exposed individual is sufficiently large, it reduces the exposed individuals and increases the isolated individuals tremendously.

Fig. 3: Shows the effect of γ_1 on the infected undetected population. It shows that when the detection rate of undetected individuals due to contact tracing is very low, the infected undetected individuals increases tremendously which will cause the total amount of infected individuals to increase in that population.

Fig. 4: Shows the effect of γ_2 on the infected detected population. It shows that the higher the rate at which infected detected individuals move to isolated individuals due to isolation techniques, the lower would be the infected detected individuals in the population.

Conclusively, since the contact rate $(\beta_1, \beta_2, \beta_3)$ plays a very vital role in the spread of the disease in the population, it is strongly recommended that to reduce the spread of the disease the detection rate of infected undetected must be sufficiently large and isolation of the Exposed and infected population together with increase in safe burial rate of the dead bodies can lead to the eradication of the disease in the population. In further research, it is necessary to look into the Sensitivity analysis and Bifurcation analysis to gain more insight to the spread posed by the disease in the population.

Competing Interests

Authors have declared that no competing interests exist.

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ISBN: 978-1-63248-075-0
DOI: 10.15224/ 978-1-63248-075-0-41

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